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DEPRESSION FOLLOWING BRAIN TRAUMA IS ENHANCED IN PATIENTS WITH MILD DISCREPANCIES BETWEEN INTELLIGENCE AND IMPAIRMENT ON NEUROPSYCHOLOGICAL SCORES[1]

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Summary.—Analysis of the MMPI (Minnesota Multiphasic Personality Inventory) scores from 135 (20 years to 60 years old) patients who had sustained closed head injuries supported the hypothesis of a nonlinear relationship between the severity of depression and the magnitude of the discrepancy between intelligence and neuropsychological proficiency. Although the MMPI Depression T scores for all groups of patients were elevated ($M = 78$, $SD = 13$), patients with the least and greatest discrepancies between intelligence and neuropsychological proficiency scored lower on Depression than patients with discrepancies within the z-score ranges −2.0 and −1.1. The results of symmetrical covariance for either depression or complex partial epileptic-like experiences before comparisons between groups suggested depression and the epileptic-like experiences share the same source of variance

Psychological depression frequently emerges months to years after brain traumas. Although the contemporary presumption is that depression is primarily due to hypometabolic activity within the prefrontal cortices or anterior cingulate, enhanced activity within the limbic structures of the temporal lobes could evoke a relative prefrontal hypofunction that would be manifested as psychological depression in conjunction with complex partial epileptic-like experiences (Persinger, 1995). The capacity of clinicians to discriminate the different neuroprocesses for depression is essential for optimal intervention. For depression due to limbic hyperactivation antiepileptic drugs or complex magnetic fields that raise neuroelectrical thresholds might be more effective than tricyclic antidepressants or selective serotonin reuptake inhibitors.

The working model (Baker-Price & Persinger, 1996) is that moderate to severe discrepancies between intelligence and neurocognitive proficiency are more likely to involve generalized brain hypofunction while mild to moderate values are more likely to reflect the consequences of smaller, time-varying dysfunctions within multiple regions of brain space. The latter heterogeneous pattern would also encourage complex partial epileptic-like signs due to phasic elevations of activity within limbic and mesiobasal temporal lobe structures that would exacerbate the depression. Consequently, the mag-

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nitude of depressive symptoms should be more evident for patients who display the intermediate cognitive-neuropsychological discrepancies rather than the minimal or maximal values.

To test this hypothesis *T* scores for the scales from the Minnesota Multiphasic Personality Inventory-168 (Vincent, Castillo, Hauser, Zapata, Stuart, Cohn, & O'Shanick, 1984) and the total score for Roberts' (Roberts, Gorman, Lee, Hines, Richardson, Riggle, & Varney, 1990) inventory for the Epileptic Spectrum Disorder were obtained for 135 successively assessed patients. The 68 men and 67 women were between 20 and 60 years of age ($M = 35.7$, $SD = 10.1$), had sustained an impact of significant mechanical energy to the head, and had been referred for a neuropsychological assessment because of subsequent problems with adaptation. Their demographic characteristics were years of formal education ($M = 11.9$, $SD = 2.6$), WAIS–R Verbal IQ 88 ($SD = 10$), Performance IQ 92 ($SD = 12$), and time since trauma: *Mdn* = 1.5 yr.

On the basis of a discrepancy score, which was the average *z* score for the seven subtests (Persinger, 1997) of the Halstead-Reitan Index minus the *z* score of the average of the Verbal and Performance Scales for the Wechsler Adult Intelligence Scale, the patients were separated into four groups. Forty-one of the patients displayed an individual, average Halstead *z* score that was more than 2 standard deviations below the individual IQ. A group of 37 patients showed discrepancies between −2.0 and −1.1 while another group of 30 had discrepancies between −1.0 and 0.00; and 27 patients had discrepancies above 0.01. To account for general elevations of the *T* scores of the MMPI, the mean score across the 10 clinical scales of the MMPI was subtracted from each scaled score (net deviation). All analysis involved SPSS software.

One-way analysis of variance with polynomial analysis supported the hypothesis. There were statistically significant nonlinear (cubic) trends between the *T* scores for Depression ($F_{3,131} = 6.44$, $p < .01$) as well as the net deviation of the Depression scale ($F_{3,131} = 6.27$, $p < .01$) as a function of the four groups who reflected the magnitude of discrepancies between intelligence and neurocognitive functioning. *Post hoc* analysis (Scheffé, $p < .05$) indicated that the patients in Group 2 (mild discrepancy) displayed higher Depression scores: $M = 85$, $SD = 12$, relative to the other three groups ($M = 75$, $SD = 13$) who did not differ significantly from each other. The average net deviation score for Group 2 was 15 ($SD = 7$) which indicated Depression scores were 1.5 standard deviations above the average of the 10 clinical scales; the other three groups (grand $M = 8$, $SD = 9$) did not differ significantly from each other. The effect size (η^2) was 10% for both measures.

A two-way analysis of variance for the mean of the *T* scores for Depression as a function of the four groups and four successive intervals of average

IQ (below 84, 85 through 92, 93 through 100, and above 101) showed no significant differences in Depression scores among groups differing in intelligence ($F_{3,112}=1.22$) but a significant difference ($F_{3,112}=4.01$, $p<.01$) for the magnitude of discrepancy. The interaction was not significant. Neither covariates for age nor time since the brain trauma, before the two-way analyses of variance, altered the significance of the effects.

There was also a significant ($p<.001$) Pearson correlation of .51 between this population's total Roberts' score ($M=75$, $SD=41$) and the T scores for Depression. Covariance first for either the scores for Depression or epileptic-like signs with the other variable as the dependent variable, eliminated ($F<1.00$) the significant nonlinear discrepancy among the groups. The last result suggests that the sources of depression and complex partial epileptic signs emerged from the same source of numerical variance and would be consistent with the explanation that phasic or intermittent elevations of activity within limbic structures could be the primary etiology of depression for this clinical subpopulation.

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